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THE MIND TWIST AND BRAIN SPOT HYPOTHESES IN PSYCHOPATHOLOGY AND NEUROPATHOLOGY

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When my friend and colleague, Professor Angier, desired that I should write a review of my general attitude toward the problems of psychopathology in their relation to the more general problems of cerebral function and of consciousness, I was minded to refuse. The point was that I considered that my ideas were little more than a mass of unproved hypotheses. However, I had just been meditating on the results of the first ten years of the Bullard Professorship of Neuropathology in the Harvard Medical School, and found myself able to draw up without great difficulty a sketch of my various unproved hypotheses, some of which I here present.

Perhaps I should preface this account of a point of view by some remarks which I hope will not be over personal. Psychologists, and especially psychiatrists, while dealing with personality day by day, are too often loath to display their own on paper. When at the Triennial Medical Congress at Washington, in 1910, I was moved in discussion to denominate two great groups of friendly opponents in the field of psychiatric theory respectively as the "mind twist men" and the "brain spot men," I was reproached by some of my best friends with making light of a grave matter. The phrases are, to be sure, of little moment; but I consider that the distinction between those who uphold the hypothesis of psychic factors as opposed to those supporting the hypothesis of encephalic factors must be drawn if we are to make any sort of progress in genuine psychopathology. The ardent parallelists (among whom, I must confess, I should not like to be numbered) would, I suppose,

say that mind twists and brain spots are all one, since everything depends upon the aspect from which one works. The methodical purist might indeed assert that he who dealt in mind twists should not commingle therewith any data concerning brain spots; and the anatomist would be sure to resent a commingling of the psychic with his own localizations. For my part, without any stringent proof, I feel that somehow the hypotheses which for better or worse I was fain to describe as the mind twist and brain spot hypotheses are in some sense and in the long run identical hypotheses. I have indeed endeavored to give expression to the concept of their essential identity in a paper entitled, "Psychopathology and Neuropathology: the Problems of Teaching and Research Contrasted,"[1] and I pointed out how pernicious in research may be the dogmatic insistence on the doctrine of psychophysical parallelism in medical and premedical courses in psychology, pernicious because it inhibits the free interchange of structural and functional concepts and the passage to and fro of workers in the several sciences. I went on to show that psychology and physiology have more in common than either has with such structural sciences as anatomy and histology, and that the main common element of both mental and cerebral processes is the time element as against the space element of the structural sciences. On this ground I further conceived that the mind twist and brain spot hypotheses for the explanation of certain forms of mental disease are entirely consistent with each other, since from a different angle each is dealing with the same facts.

My point of view here is not quite naïve or quite so innocent of metaphysical speculation as the anatomist often pretends to be. The attitude in question is one strongly influenced by my work for some years past in Professor Josiah Royce's Logical Seminary, in which the fundamental concepts of science have been taken up. I should not wish, however, to convey the impression that Professor Royce is in entire agreement with my point of view.

Any logician must, however, be readily convinced that the current classroom distinctions between organic and functional disease, especially between organic and functional nervous diseases, are flimsy distinctions. They often amount to saying that a disease shall remain functional only so long as the microscope or other technical tool shall fail to prove their organic nature. Such distinctions may be practical; I have even heard them termed prag-

[1] *American Journal of Psychology*, 1912, 23.

matic, although I doubt whether the true pragmatist could see much use in the distinctions as drawn. The concepts of structure and function have also been considered among Professor Royce's varied seminary topics, and several definitions have been proposed. The most interesting of these runs to the effect that the functional among diseases is the disease which is reversible, either practically or theoretically, in such wise that the original condition can be approximately regained. It is obvious that this definition, if sound, will not jibe altogether with the one above mentioned, namely: the vague concept of the functional as that which has not yet been proved to be structural.

In securing a working definition of the functional in disease, it will always prove necessary to adopt some definition of disease itself. Two obvious lines of distinction have occurred to Royce's seminarians, which may be briefly characterized as leading to the concept of the abnormal and the concept of the morbid. The abnormal is very possibly an entirely quantitative distinction, including as its leading varieties the supernormal and the subnormal. The greatly supernormal or greatly subnormal may be termed anomalous; but anomalies are not necessarily, although they prove often to be attended by, diseases. The morbid (this term is for some reason indelicate and has been somewhat illogically replaced with the term *pathological* in modern writing) may consist in, or be produced by, the abnormal; but a deeper account of the morbid is probably to consider it as a name for conditions which somehow defeat the evolutionary use or object of the cell or mechanism in question. Thus, a condition which entails the premature death of the cell or a loss of its important appendages or organoids would be a morbid condition. Thus the concept of pathology would have at its core the teleological concept of the morbid, but would have as a rule also to consider those quantitative variations from the normal which we gather under the term *abnormal*. It is a profound, but here not especially pertinent, question how far the concept of the morbid is itself also a quantitative affair. But the main point I here make concerning the concept of the morbid is that it is a biological concept and not a broadly physical one.

I should not venture here to offer these truisms if I were not convinced that the psychologist in the academic sense seems to believe, and at any rate often leads his students to believe, that psychopathology is in some sense a science of psychical anomalies; that is, of psychic processes that are figureable in curves at the

upper or lower end of the normal curves. When the academic student begins to get a grip upon the essential problems as they are presented by a patient, or by those remains of human beings which often yield the greatest returns for a given amount of investigation, he discovers that a science of supernormal and subnormal measures leaves him entirely at loose ends and does not get him a millimeter onwards with his problem. Here, it does not do to speak with authority; yet the returns to the committee of the American Psychological Association, on which I served, as those returns were prepared by Professor Franz, indicated that those concerned with the problem of interrelations betwixt psychology and medicine were split into camps along the above lines, namely: A camp of those claiming the virtues of studying the quantitatively anomalous, and a camp of those who wish to study the biologically non-adaptive or the evolutionarily unfit.

So much by way of preaching. As a practical method of getting the students, and particularly the graduate students, to appreciate the science of the psychiatrist's problem as the pathologist sees it, I have in the last few years come to express the idea in somewhat the following terms: I first beg the student to consider the nervous system as theoretically reducible to a linear system of neurones, separated in the Sherringtonian manner by synaptic planes. I then point out that, if given muscles are convulsed as in an epileptic attack, we cannot safely state that the spinomuscular neurones which supply the convulsed muscles are in any respect abnormal, except that we must admit that their existence and participation are necessary for the production of the convulsions in question. Similarly with the corticospinal neurones, whose impulses are conceived to run into the spinomuscular neurones at the appropriate synaptic planes (under various conditions of inhibition and reinforcement which may be neglected). In epilepsy, we do not often discover that the corticospinal neurones are any more visibly diseased than are the spinomuscular neurones. In point of fact, the analysis of epilepsy, as of a great variety of neuropathological conditions, involves considering one by one, at greater and greater removes from the seat of the physical phenomenon of convulsions, the successive neurones which are indispensable in the production of the symptom but are not responsible therefor. In epilepsy, in point of fact, in the majority of cases in which science has at all made out the immediate cause of the convulsions, it has been discovered that the seat of the lesion is not in the motor neurones but

on the afferent side of the apparatus. These considerations for epilepsy I brought together in a paper entitled, "On the Mechanism of Gliosis in Acquired Epilepsy."[1] I there pointed out the relation of my own "microphysical" theory, and its endeavor to describe certain pericellular conditions which might well bring epilepsy about, to the "level" theories of Hughlings Jackson, as well as the relations of my theory to the Sherringtonian concept of synapses. In that paper I gave a highly demonstrative case of epilepsy of nine weeks' duration, terminating in four days of practically continuous convulsions due to a virtually non-destructive lesion of a sensory area. There was a focal encephalitis of the right cornu ammonis which, whatever its cause, exhibited an interstitial accumulation of neuroglia cells, leaving the nerve cells virtually intact. My hypothesis was that fresh surfaces of separation had been interposed between sets of nerve elements. I supposed that these elements, having their currents in the forward direction, and being placed under fresh conditions of intimate pressure, would initiate continuous or lasting stimuli, which would set the remainder of the apparatus moving in an abnormal fashion.

The point which I wish to make for the present purpose is that in the case of epilepsy just mentioned, and in a vast majority of cases of neuropathy of every sort, we may well suppose that the neurones which lie outside the focus of disease, and the muscles, glands, or other organs which they supply, may be entirely normal and executive of their normal functions. I tried to sum this concept up in the following phrase: Neurones may be intrinsically normal whereas extrinsically abnormal; entirely normal structures may accordingly purvey and be necessary in the production of disease.

This simple concept of the intrinsically normal yet extrinsically abnormal or morbid is of great use in psychopathology. I find it dominating my own methods of thinking. When public attention was directed, in the period just preceding the Washington Congress above mentioned, to the problem of dementia præcox, it seemed to me that very probably the brains of dementia præcox patients would be found to be normal; at least it was true that some of the most eminent psychiatrists had been unable to discover, in the majority of cases of mental disease (in which field dementia præcox must largely bulk), anything abnormal, let alone morbid. Although it had become a household word that insanity was brain disease,

[1] *American Journal of Insanity*, 1908, 64.

yet there was little or no evidence or hope that the brain disease would be soon discovered. Under this assurance, I wrote a section of my paper on dementia præcox entitled, "A Study of the Dementia Præcox Group in the Light of Certain Cases showing Anomalies or Scleroses in Particular Brain Regions."[1] I there pointed out that the disease diabetes mellitus, being distinguished among other diseases by the production of great amounts of urine with an abnormal amount of sugar, might well be conceived by the tyro as a kidney disease. Yet upon investigation, it turns out that the lesions of the kidney in diabetes mellitus are negligible and inessential, and that the disease itself must be related to remote or unknown organs. This analogue points the way to a broadening of the concept of the intrinsically-normal-but-extrinsically-abnormal to include other elements than neurones, and indeed to include the chain of organs which we latterly suppose are concerned in the production of internal secretions. Just as the intrinsically normal kidney is extrinsically abnormal in diabetes mellitus in the sense that it purveys a large amount of sugar in the urine, so *might* the brain in dementia præcox be intrinsically normal yet extrinsically abnormal, in the sense of producing delusions, catatonic excitement or stupor, or other characteristic symptoms whose genuine origin might conceivably lie entirely outside the nervous system.

This was my conception of the probabilities with respect to dementia præcox when I entered upon the study of a series of brains in that disease in preparation for the Congress of 1910. It still remains my conception of conditions in the sister disease, manic-depressive insanity; but in dementia præcox I was greatly surprised to find that the vast majority of cases were distinguished as to their brains by the possession of distinct though mild lesions in the nature of anomalies, atrophies, or scleroses, which in so labile an organ as the brain must perforce have their effects upon brain functions.

This long preamble is probably justifiable in preparation for showing why a psychopathologist should find himself a localizer despite logical predilections against brain localization for psychic processes. I may briefly state the view to which my as yet unproved hypotheses seem to be leading, as follows: It seems to me that just as a tremendous leap forward was taken when the Flourens view of the interchangeability of brain parts was sup-

[1] *American Journal of Insanity*, 1910, 67.

planted by a roughly localizing view, and when the bilaterality of brain function began to be unravelled, along with the data showing the seizing of some functions by one hemisphere as against the other, so we may be now in the process of a great advance as we come to a full recognition of the value of distinguishing the parts of the cortex which lie forward of the fissure of Rolando and above the fissure of Sylvius from the parts which lie behind and below those fissures. For it seems to me that the indications are strong that the silent portions of the pre-Rolandic areas of the cortex, forming the anterior association center of Flechsig, are predominantly motor in function; whereas the correlative backward-lying association center is predominantly sensory. Just as it is convenient at some times to divide the earth into an eastern and a western hemisphere, and sometimes into a northern and a southern hemisphere, so it may be well for many purposes to distinguish the left hemisphere of the brain from the right, but for other purposes it may turn out that the pre-Rolandic and supra-Sylvian portions of both hemispheres, with their concomitant commissural fibers in the corpus callosum, should be fairly sharply distinguished from the post-Rolandic and infra-Sylvian regions of the cortex. It is true that the forward "hemisphere" is of far less bulk than the rearward and nether "hemisphere"; but this difference in size is only another illustration of the difference which holds throughout the nervous system between the afferent and efferent fiber systems which compare quantitatively always much in favor of the afferent.

It does not appear that the theoretical distinctions which are possible between these two portions of the cortex have been developed as elaborately as they should be by either the anatomists or the physiologists or the pathologists. I have personally been led to wonder whether there is any basis for considering the pre-Rolandic tissues as having anything whatever to do with consciousness, that is, with *consciousness in its cognitive sense.* This was the burden of my communication at the New Haven meeting of the American Psychological Association in the closing days of 1913.

I arrived at this idea in a concrete fashion. I found in the course of my anatomical analysis of dementia præcox brains that cases with frontal lesions were chiefly cases distinguished by the possession of delusions; that is, belonged to the so-called paranoid group of dementia præcox (to employ Kraepelin's 1899 classification). I found that the catatonic cases were correlated, not so much with pre-Rolandic atrophies, as with atrophies of the post-

central, parietal, or in some cases cerebellar, tissues. This seemed at first glance a surprising correlation, since delusions are apparently of a psychic texture, whereas the muscular spasms, inhibitions, stereotypies, and impulsivities of catatonia, suggest the efferent rather than the afferent system. Upon reflection, however, it appeared that a similar apparent difficulty lodged in the sensory basis of many epilepsies, as alluded to above. After all, it was not the content of the delusions which was so important to the patient; it was the process or formation of these delusions. It was not so much the false beliefs with which either society or the patient himself was concerned; it was rather with the maintenance of the falsely believing process, the morbid will to believe. Every one's working day is a kaleidoscope of false beliefs. But luckily they correct themselves or get supplanted in such wise that a normal attitude ensues. The psychopathology of insane delusions was consequently to be interpreted rather as a psychology of false believing, and was better conceived as a matter of behaviorism than of introspective psychology. Thus, whether my anatomical correlations were sound or not, I was able to arrive at an interesting concept of delusions as a form of conduct rather than as a form of static mental contents.

On the basis of this concept, I was led to analyze delusions of the various groups, classified (as we had by chance chosen to classify in the Danvers symptom catalogue) according to Wernicke into autopsychic, allopsychic, and somatopsychic. I quickly found that somatic delusions are far more representative of actual visceral conditions than is usually held. It is accordingly possible to conceive of many somatic delusions as virtually illusory in nature. Conclusions in this direction were published in a paper "On Somatic Sources of Somatic Delusions."[1]

This led to a study of allopsychic delusions, namely: those false beliefs dealing with the environment and especially with the social environment, which was published with A. W. Stearns in a paper entitled, "How Far is the Environment Responsible for Delusions."[2] The majority of these cases were found to be more truly instances of autopsychic or personal delusions, than environmental. This study was followed by one on the correlation between delusions and cortex lesions in the pronouncedly organic disease general paresis.[3] Incidentally, we here again found that the in-

[1] *Journal of Abnormal Psychology*, 1912–1913.
[2] *Journal of Abnormal Psychology*, 1913.
[3] With A. S. Tepper, *Journal of Abnormal Psychology*, 1913.

stances of somatic delusions sometimes complicating the picture of general paresis were usually attended by an adequate peripheral basis. Thus, a patient who described himself as blind but as having a filter over his eyes in such wise that he could see, turned out to be the victim of cell losses in the visuo-psychic type of cortex, with maximal pigmentation of the neuroglia cells. The patient should have expressed his delusion by saying that he could see but had a cortical veil preventing his perceiving properly.

More important, however, was the discovery that autopsychic delusions and that characteristic ruin of personality which we classically assign to general paresis must be correlated with frontal lobe lesions. In the non-autopsychic group, we found the lesions distributed elsewhere than in the frontal region; that is to say, we found these non-autopsychic cases failing to show the classical frontal brunt of the distinctive process. Here, then, was concrete evidence that the personality, conscious as it seems, was more closely related with the pre-Rolandic than with the post-Rolandic tissues; with the efferent mechanism more than with the afferent mechanism. It seems to me that here again we are securing evidence which supports to some extent the objectivistic or behavioristic trend in modern psychology. It seems possible that psychopathology, even in the exquisitely psychic fields of the delusions, will not gain so much by an endeavor to ferret out the innermost psychic secrets of the patient as by a careful quantitative study of his reactions in the line of conduct. If some method could be devised for obtaining the survival values of these actual processes of conduct rather than their academic quantitative values, we should be so much further on the road to a behavioristic psychopathology.

A similar line of thought follows suit in respect to catatonia. This exquisitely muscular phenomenon, like the similar phenomenon of epilepsy, turns out, as it seems to me, to be more a sensory than a motor affair. Just as delusions had less sensorial significance than they had significance on the side of action, so the catatonic and cataleptic phenomena turn out to have less significance from the side of action than from the side of the sensorial, or at all events the afferent, apparatus. Again discounting the question whether the anatomical correlations in dementia præcox upon which I founded this idea are sound or not sound, it is clear that some heuristic value must fain attach to this concept. It is, in any event, important to consider how far catalepsy is actually due to a sort of morbid kinæsthesia. Suppose a postcentral disease which should

provide to the surfaces of separation between neurones their initial stimulus, purveying as it were quasiperipheral stimuli of a given kinetic quality, then perchance the remainder of the general mechanism although quite normal (*intrinsically*) would have to react in the cataleptic way. What might seem to be a will disturbance, or a disturbance initiated in or near the precentral gyrus as a form of abnormal or morbid conduct, might perchance be executed by a thoroughly normal precentral and frontal mechanism on the basis of abnormal or morbid conditions in the post-central region. Upon this basis might be built up, in short, a kinæsthetic or quasikinæsthetic theory of catatonic and cataleptic phenomena.

An illustration from less disputed fields may serve to bring out the point. It is well known that experimental physiology has shown that there are two centers for conjugate deviation; namely, a center in the angular gyrus, which lies posterior to the Rolandic fissure, and a center in the middle frontal gyrus which lies anterior. Should we regard the results of stimulating these two areas as entirely similar? Are we to suppose that projection fibers run directly from both these cortical areas to the appropriate oculomotor neurones? Are we not rather to suppose that they stand to one another in some logical sense resembling that in which the area of Wernicke for sensory speech stands to the area of Broca for motor speech? Can we perhaps generalize that many or the majority of the complex functions for which the cerebral cortex is built are thus doubly supplied fore and aft by mechanisms which on the one hand are more closely related to conduct or behavior elaboration, and on the other hand to kinæsthetic or cognitive elaboration?

This leads me to quote with as much disapproval as I becomingly can from Wundt's expression of his anti-localizing views in the first volume on speech in his *Völkerpsychologie*. Wundt decries the conception that every cortical brain cell harbors some idea. The unregenerate physiologist, according to Wundt, holds the conscious conception that deposits of different ideas or thoughts are distributed over the cortex in districts; one for sound impressions, one for visual images, etc. These compartments of the cortex, according to Wundt, were conceived by illogical physiologists as in part occupied by ideas, and in part engaged ahead for future occupants. Destruction of a center for ideas would of course destroy ideas deposited; but fortunately these destroyed ideas could be replaced by new ones occupying cells now vacant. Such a restoration of function would not differ essentially from the process

of normal brain development so long as still disengaged cells remained available. Prior to the work of Broca, in 1861, according to Wundt, every one had thought vocal sounds to be of physical origin but words of psychic origin. A word, to be sure, required the physical aid of sound production, but nevertheless every word was really the outcome of a concept and was exactly as much a psychic affair as desire or will; but Broca showed that motor (or better, according to Wundt, ataxic) aphasia depended on lesions of a certain part of the brain (inferior frontal convolution). Wernicke followed in 1874 by showing that sensory (or better, according to Wundt, amnestic) aphasia depended upon lesions of a certain other part of the brain (superior temporal convolution). Then followed Kussmaul's scheme in 1877, which as modified by Lichtheim in 1885, seemed to show that at least thirteen different kinds of aphasia might be produced by lesions appropriately placed in different parts of the brain. Then followed the work of Meynert and the work of Munk.

I suppose there can be no objection to this schematic account of the history of the doctrine of aphasia as developed by Wundt, but I should now wonder whether it is advisable to consider that any idea, or at all events any cognitive process, can or ought to be related to such an area as that of Broca. The area of Broca, like the area near by for conjugate deviation, or the so-called graphic center or similar congeries of interrelated elements, may be supposed to be, or to take part in, a synergic mechanism for one or other purpose. The frontal part of the brain is doubtless full of these synergic mechanisms. The negation of personality entailed by frontal lobe disorder indeed indicates that the synergic mechanisms, or kinetic patterns or schemata, normally contained in the anterior association center are even capable of novelty production, of the faculty of innovation, upon which our title to supremacy as human beings depends. Accordingly, I should wonder whether the analysis of the effects wrought in such areas as that of Broca was not more a matter of behavioristic psychology than of introspective. Some might inquire whether it were not well to consider such an area as entirely physiological in its action. To this form of expression, I should have no objection if it be understood that in some way or other we must explain the correlation of personality with these forward lying cell systems. It is probable that these cell systems of the anterior association area are every whit as much entitled to psychological consideration as the cell systems of the

posterior association center. Yet the operations of the latter are very possibly on theoretical grounds far more open to introspective study than are the operations of the forward lying cell systems.

Naturally, the products of the action of the precentral gyrus or of the area of Broca do get representation on the cognitive side, that is mainly in the kinæsthetic manner and doubtless more back of the fissure of Rolando than forward thereof. On this account, introspection has been an important, or even essential, method in the analysis of behavioristic problems, since the kinæsthetic or other similar record of what is being done will often serve as the best guide for the actual course of events when the behavioristic method itself may not yet be able to cope with technical difficulties. On the other hand, after all, what the psychopathologist as well as the psychologist wants to register is the acts and deeds,—that is, the conduct,—of the individual, and on this account the operations of the anterior association center of Flechsig are of prime value.

In partial support of these ideas, I have recently studied the literature and certain casualty ward records with post-mortem studies available to me, with the object of learning how far what we term clinically consciousness and unconsciousness are consistent with extensive lesions of different parts of the cortex. Already the Crowbar Case and other similar cases stood to prove that the frontal cortex might be seriously injured without permanent impairment of consciousness in the slightest degree. An affection of character might well ensue, indeed is described as having ensued in the crowbar case; but it does not appear that consciousness, or at all events selfconsciousness, was lastingly injured in that classical case.

The considerations of this latter study led me to consider the meaning of the term consciousness. It seems to me that the term should be restricted to what it etymologically seems to signify, namely, cognition and compounds of cognition. It seems to me that the components of will and possibly those of emotion are entirely, or almost entirely, gotten into what we call consciousness by the cognitive route of kinæsthesia, and that there is great question how much elementary introspective stuff there is to the will and the emotions which cannot better be accounted for on the basis of kinæsthesia. If this account of consciousness as in a sense cognitive is a good simplification of nomenclature, I would suggest that a similar simplification in the field of the so-called unconscious is sadly needed. Some textbooks on psychology

seem to identify consciousness with mind. The unconscious is, according to these authors, surely much more than the non-conscious, and indeed has been hypostasized into a novel and mystical entity having all the old warmth and intimacy of the so-called conscious and many strange intimacies besides. If one tries various current definitions of the unconscious by replacing the term *unconscious* by such a term as *non-conscious* or *non-mental*, one discovers how much balderdash has been inflicted upon us by many exponents of mystery.

So much will suffice for a doubtless far too personal and over dogmatic account of my reaction to the present situation in psychopathology and psychology. I do not vouch for the ultimacy of any of the ideas expressed, and must place upon my friend, Professor Angier, all responsibility for the premature delivery of possibly non-viable children of fancy.

Summary.—I am sure that some of the dozen or more separate conceptions to which I have asked attention in the above review will hardly carry conviction in the present sketchy form.

1. The *mind-twist versus brain-spot hypotheses* have nowhere been discussed *in extenso* (although see articles on "The Problems of Teaching and Research Contrasted and a Study of the Dementia Præcox Group," etc., mentioned in text), and I am not sure that the distinction will strike the reader as more than a fresh sample of psychophysical parallelism. Without special title to a viewpoint, I wish however to say that personally neither parallelism nor interactionism seems to me safe ground and that some kind of identity hypothesis for all the operations concerned would be better consonant with my views. One thing will be clear from the above sketch, viz., that it may well be possible that mental operations of the introspective kind are not correlatable (in any sense) with a good part of the operations of the cerebral cortex.

2. The definition of *consciousness as equivalent to cognition and compounds of cognition* leaves the non-cognitive portions of the mind (will and emotions) only capable of introspection by the kinæsthetic and allied sensorial routes. But, whether the above definition is correct or not, it is at least clear that many authors in the past have confused the issue by identifying mind with consciousness, at a stage when neither concept was capable of exact statement.

3. The pathological evidences which have absorbed my personal attention have led me to a *reëmphasis of the Flechsig concept of*

anterior and posterior association centers, to a natural correlation of consciousness and the entire sensory portion of the mind with activities of the posterior association center, and to a similar correlation of non-conscious, *i. e.*, objectivistic or behavioristic portions of the mind (notably the voluntary faculties) with activities of the anterior association center: the *prepallium* (pre-Rolandic cortex) would thus be more closely related with behavior (kinetic and pragmatic schemata) and the *postpallium* (post-Rolandic and infra-Sylvian cortex) most closely related with consciousness.

4. But, if the *prepallium* is more an organ of behavior than the receiving *postpallial* mechanism, it is expressly to be stated that the capacity for novelty-production, or innovating power, is not to be abstracted from the prepallial neurones. Such innovating power, exquisitely mental as it seems, is not necessarily conscious in the sense of essentially cognizable. It is perhaps only the *history* of our innovations and inhibitions which we register in the postpallial mechanisms. Arguments in this direction are to be drawn from the decisive ruin of the personality which attends *prepallial* destructive processes in general paresis of the insane.

5. A sketch is offered to show that the non-conscious, *i. e.*, non-cognitive, side of delusion-formation is perhaps more important than the conscious (or contentwise) side. At least the morbid correlates of delusion-formation seem to be prepallial rather than postpallial disorder as a rule.

6. The reverse seems to hold for such apparently motor or behavior phenomena as epileptic and cataleptic phenomena: these are possibly based more often on postpallial (sensorial?, kinæsthetic?) disorder than on intrinsic disorder of behavior mechanisms.

GENERAL REVIEWS AND SUMMARIES

THE FUNCTIONS OF THE CEREBRUM[1]

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The study, or science, of cerebral function is, like many other sciences, in a complex stage of its development. The relation of the brain to other parts of the body or to mental processes is no longer considered to be a mysterious matter, nor are the simple explanations of its functions believed to be entirely satisfactory. The collection of facts has proceeded to such a point that it is now generally recognized that even the relations of the cerebral cells to the production of movement are not to be explained in a simple manner. Many observations of motor, sensory and association disturbances accompanying cerebral lesions have been recorded which can not be explained by the older hypotheses, and which, in fact, negative them. Some have been insisting for a number of years that the cerebral relations are not simple, but because of certain practical needs and applications, the discrepancies have been largely disregarded. It must be understood that cerebral relations are not becoming complex in the sense that more areas with definite functions are being discovered, but in the sense that many functional variations are being recorded, that there are many so-called anomalous disturbances associated with cerebral lesions. For this reason the simple explanations or theories are no longer acceptable, and many which have hitherto been considered almost like statements of fact are undergoing radical modifications.

The general belief that aphasias in right-handed people are produced exclusively by lesions in the left hemisphere and in left-handed people by corresponding lesions in the right hemisphere is not borne out by a number of cases, and it is valuable to have the additional negative case which has been described by Long (6). On autopsy, Long discovered in this left-handed individual that

[1] For last previous review see PSYCHOL. BULL., 1913, 10, 125–138.

the aphasia was associated with lesions of the second and third frontal convolutions, of the island of Reil, of the internal capsule, and of the surrounding regions in the left hemisphere. The reason for an association of such contralateral lesions (*i. e.*, not corresponding to the hemisphere of greater voluntary control) with the speech disturbances is not plain, but it is apparent that in this case, and probably in others, there may be an anomaly in the motor and sensory association functions of the cerebrum, or, which seems to the reviewer more probable, that we have in this case only an example of the normal variation in function in different individuals.

The symptom variations which are found in individuals with lesions in the occipital lobes are also very great. Certain cases have been described in which very extensive lesions have not been accompanied by a complete loss of vision, while in other cases comparatively small lesions resulted in almost complete loss. At other times it is found that even in apparently complete bilateral lesions some visual ability remains. In extensive calcarine lesions, and especially in those cases in which there are visual defects for special retinal segments, it is not unusual to find that patients still retain certain visual ideas and have visual experiences which are of the nature of hallucinations. In the case which has been described by Josefson (5), this was found. In this patient, in addition to the general mental changes associated with the pressure, etc., due to the cerebral lesion, which was produced by a glioma or a sarcoma in the left occipital lobe, there were found homonymous hemianopsia with optical aphasia and alexia, but the patient had marked visual hallucinations in the right visual field corresponding to the hemianopic area, and the hallucinatory figures were at times larger than normal. Such cases have more than the particular interest for the purpose of localization of cerebral function, for they indicate that even though there be large amounts of destruction of cells and fibres, the mental functions, supposedly connected with the activity of these cells and fibres, may still persist. They also indicate how little we know at present of the relations of cerebral cell activity and such mental things as ideas.

Minkowski (9), after extirpating the occipital regions of the cat's brain, and in certain cases the eyes, has determined the paths of degeneration, and especially the relation of the degenerations in the external corpus geniculatum due to lesions of the striate area (calcarine cortex). His results indicate that the external geniculate body is the only subcortical structure in which, following

an extirpation of the striate area, a degeneration of the ganglion cells is determinable, for neither in the pulvinar nor in the anterior quadrigeminal body was he able to determine accurately that there were pathological changes in the ganglion cells following such injuries. These latter structures, pulvinar and quadrigeminal body, do, however, show degenerations following the destruction of cortical areas near the striate area. Minkowski has also been able to demonstrate a finer anatomical relationship between the calcarine cortex and the geniculate body, for he has observed that the anterior portion of the striate area is connected largely with the anterior portion of the external geniculate body and the posterior portion of the visual cortex with the posterior part of that ganglion. A somewhat similar relationship is found to exist between the eyes and the corpora geniculata, and Minkowski has been able to show that there is a fair degree of correspondence, or anatomical relationship, of the retinal areas to parts of the geniculate bodies.

Boyd and Hopwood (3) have reported the autopsy findings and the clinical phenomena in an interesting case, which, by exclusion, gives valuable information regarding the more definite localization of the cerebral center for hearing. In their patient they discovered in the left temporal lobe of the cerebrum a large cyst which destroyed the superior and middle temporal gyri, with the exception of the anterior portions. In this patient no impairment of hearing was discovered by the clinical examinations, and the evidence appears to be conclusive that the portions of the temporal lobes which were destroyed are not primarily, or necessarily, concerned with the perception of sounds. It will be remembered that Campbell, on histological ground, has laid particular stress upon the anterior gyrus of Heschl as a special sensory center and as being closely associated with the function of hearing; in the patient described by Boyd and Hopwood this gyrus escaped involvement, and the authors conclude that the results tend to support Campbell's conclusion that only the anterior parts of the superior temporals are hearing centers.

It is well known that in certain animals, as the cat and dog, paralyses due to cortical destructions are not permanent, the destruction of the motor area resulting in only a temporary paralysis which is followed by a subsequent practically complete recovery of control of voluntary movement in the involved segment. It has also been believed that after the destruction of part of the motor area in man and in the higher apes the paralysis resulting from the

lesion is permanent. In a chimpanzee, however, Brown and Sherrington (4) destroyed the left cerebral arm area, and although this area was completely destroyed, voluntary movements at the right elbow could be performed, and in a comparatively few months such a great amount of return of voluntary movements of the whole arm, took place that eventually there was no obvious difference in the control of the movements of this arm as compared with the left arm, which was normally innervated. The authors conclude that this return of function can not be due to a regeneration of the area which had been destroyed, nor to the assumption of this function by the corresponding portion of the right hemisphere, nor to the vicarious function of the left postcentral cortex. Following extensive precentral cortical lesions in man, there may be immediate extensive paralyses, even hemiplegias, which are expected to, and do, show a certain amount of improvement in the weeks succeeding the initial cerebral insult. Whether the partial restoration of functional control is due, as has been assumed by many, to a subsidence of the pressure, or to some conditions as yet unknown can not be definitely solved. The results of the experiments of Brown and Sherrington are, however, suggestive that even though there be a destruction of the cells, there may follow a functional amelioration. The possible ways in which this may be brought about are numerous, and we need only mention, by way of suggestion in this connection, the well-known efferent functions of the caudate and lenticular nuclei and of the cerebellum. In this connection also, it may not be amiss to call attention to the fact that the reverse condition has sometimes been met with, *viz.*, the appearance of organic-like paralyses without corresponding lesions of the cerebrum or of the other parts of the nervous system. Scientifically, it is neither satisfying nor exact to call these latter conditions hysterical or functional, for they tend to show that the cerebral relations are not as simple as they have been conceived to be, and the use of a special name is, in this case, only an indication of lack of knowledge.

Romagna-Manoia (12) has, in his book, collected very completely the phenomena associated with hemiplegia in its different stages, and has given us a careful account of minute experimental results on this matter, which are most interesting for the purpose of diagnosis, but which, in addition, are of value for the understanding of the relations of the cerebrum to other portions of the nervous system. The facts are too numerous to be detailed in the present

brief review, but it may be mentioned that the reflexes, the resulting contractures, and like phenomena accompanying the various paralyses have been carefully described and amply dealt with.

An interesting study of the stages of development of the cortex of an eighteen weeks' human embryo has been reported by Bolton and Moyes (**2**), who give us an account of the cell structure which, in its way, is a companion research to those of Flechsig on the myelinization of the brain. The embryo brain which is described contained only beginnings of the Rolandic, calcarine and parieto-occipital (temporary) fissures, the cingulum and the opening of the island of Reil. The Betz cell area was well marked and easily localized, although the authors say that the discovery of the Betz cells in a fetus of this age was a surprise to them. This area, probably on account of the lack of infolding of the central fissure, has a much wider extent than that in the brains of newborn children and adults, and in general a similar statement may be made regarding the calcarine type of cortex. Much of the cortex showed comparatively slight development, although the precentral and postcentral areas are remarkably well evolved in comparison with the remainder. They note, and in this there is support for Bolton's previous contentions, that the anterior frontal or prefrontal cortex is throughout its depth extremely embryonic in structure. No systematic attempt has been made, as did Flechsig, to correlate the findings with functional development, but it is apparent that the greater development of the precentral and postcentral areas, at this stage of its life, is to be correlated with the well-known activities of the embryo. It is believed, the authors conclude, that this development "bears a definite relation . . . to the already stable condition of the lower reflex mechanisms."

By histological methods Malone (**7, 8**) has attempted to differentiate the types of cells which are concerned with different types of function, and for this purpose he has examined a number of parts of the cerebrum and has applied his methods especially to the examination of definite cell groups. In the vagal nucleus, he shows, there are three types of cells which may be taken as correspondents of the three kinds of muscle (striated, the heart, and smooth), which are innervated by the cells in this nucleus. He reports that "there is no gradual transition in structure between the cells of the afferent and motor chains, and there is no indication of the beginning of motor structure in afferent cells. Those cells in the efferent chain whose function consists exclusively or primarily in conducting

impulses through the chain to cross striated muscle, or between motor centers, are characterized by a common structure, which differs according to the position of the cell in the motor series." He furthermore remarks that "an important field is open to students of the central nervous system in studying the cell structure of different cell groups, and in correlating a definite structure with a definite cell activity wherever this is possible"; and he concludes that "a definite type of cell structure corresponds to a definite cell function." If we consider this "conclusion" as a "point of view," we should be more nearly correct, for it has not been demonstrated that cells of different sizes and of slightly different appearances may not have similar function. If it could be shown that all kinds of histologically different cells in the cerebrum differ in function, and that each different combination of cells (cytoarchitecture) also represented a different function, the problem of the cerebral physiologist would become, if not less arduous, at least better defined. The connections of cortical cells with extracerebral parts and the interconnection of cortical cells must be investigated before we shall have definite knowledge of the function of special groups or groupings in the cerebrum, but for many it is more satisfying to speculate how things may be, rather than to investigate how they are. It is because of this failing that we possess many assumptions and hypotheses regarding the functions of individual cells. We should be more scientifically served if there were published fewer speculations and more serious attempts at anatomo-functional correlations, such as that of Malone.

During the past ten years the corpus callosum has been the object of numerous pathological and clinical studies. This has been due principally to the fact that lesions in this region have resulted in various types of apraxia and because it appears that by means of this structure the two hemispheres are closely connected. Our knowledge, however, of the course of the fibers which make up this structure has been rather inadequate, but van Valkenburg (13) has now compared the results of animal experiments and pathological human material, and although he has not been able to fix a definite relation for the callosal fibers for all portions of the brain, it is apparent that these fibers do pass from one hemisphere to the other and apparently bind corresponding or allied areas. In this work he shows that in certain parts the callosal fibers probably arise from the fifth and the sixth layers of cortical cells and that in proceeding to the opposite side they terminate in

the third and, possibly, the fourth cortical cell layer. He has pointed out another interesting fact, viz., that there is a connection between the precentral area of one side and the opposite postcentral area. The other extensive connections, between the corresponding occipital lobes, and between the corresponding frontal lobes, are better known, on account of the association of lesions in these tracts with the phenomena known as apraxia. The importance of a knowledge of these tracts is now being more appreciated, and we may hope to obtain in the not distant future more information of the corresponding areal connections of these tracts of the callosum and the processes or functions which are subserved.

Of a somewhat similar anatomical nature, and with similar physiological value, is the study of Besta (1), who has carefully worked over the paths of degenerations following various lesions in the brains of dogs and cats. By comparing the results of the degenerations of fibers by the Marchi method and those of cell atrophy, he has determined the paths of fibers and, consequently, of the impulses which normally pass between the cerebrum and cerebellum. One of the most interesting facts in his results is that, contrary to the contentions of von Monakow and others, he did not find, and because of this he denies that there are any cerebropetal fibers in the pedunculi cerebri.

In a general article, Pawlow (11) has reported his views of higher nervous function which have resulted from his experiments on psychic secretions. The psychic secretions which he has been mainly instrumental in demonstrating in animals he has termed "conditional reflexes," since, unlike the organic or the well-established reflexes which are carried out by the spinal cord and the medulla oblongata, they may be affected by and they depend upon a multitude of conditions. It is well known that when food is presented to an animal so that the animal sees the food, secretion by the digestive glands begins before the food is taken into the mouth. Pawlow has also shown that if, at the time food is presented, a stimulus which is extraneous to this food be also given to the animal, the combination of stimuli results in a secretion. After this combination, food plus an extra stimulus, has been used with an animal a number of times, it becomes sufficient for the production of the digestive secretions to present to the animal solely the extraneous stimulation which has been previously associated with the food. The non-food stimulus then gives rise to, or is accompanied by, the reflex activity. It has also been possible to show that even

though the character of the extraneous stimulus be changed, this non-food still results in secretion production. For example, if at the times that food be presented an electrical stimulus be given to the skin of the foot, the secretion of saliva becomes associated reflexly with this electrical stimulus. After the reflex has been well established, it is no longer necessary to present food to bring about the reflex, nor is it even necessary to stimulate the foot by electrical means; painful stimuli, such as cutting and burning, are also accompanied by the same sort of reflex activity, viz., secretion. Because of these and many other facts Pawlow points out, or concludes, that part of the function of the central nervous system, and especially that of the cerebrum, is that of dispersion, not only that of correlation or integration, of impulses, and that the cerebrum acts to a great extent, as the reviewer pointed out more than ten years ago, for the production of new types of activity and new reflexes, and, perhaps, anatomically for the purpose of making new connections.

The localization of functions in the cerebral cortex, or in the brain of man, von Monakow (10) writes, constitutes a complex problem, which is the goal of localization in the central nervous system in general. He points out, as others have previously done, that localization may be considered in three different ways: first, the localization in an anatomical sense, viz., groups of fibers and cytoarchitectonic; secondly, the localization according to symptoms and symptom groups, or the correlation of irritative or defect phenomena with localized injuries to the brain; and thirdly, the definite localization of mental functions. These three ways of looking at cerebral functions are frequently, perhaps usually, not differentiated. It is, however, important and necessary that they shall be kept clearly in mind if we are to draw a distinction between what may be called the "association of cerebral lesions with motor, sensory and association defects," and the "localization of mental processes." Every cerebral injury results in two stages or forms of symptoms, the initial and the residual. Both of these must be carefully noted and analyzed if we are to understand the complexity and the integration of cerebral activities. Among the most important of the general effects is that of diaschisis, which results in a cessation of impulses over the whole cortex and which is essentially a "reduction or cessation of the capacity for excitation." Widely separated areas are always involved even when only a very small portion of the cortex is affected. Some destructions may give rise

to perfectly obvious defects or exaggerations, others may give rise to effects which are to be determined only in an indirect manner by a process of exclusion, while others again appear to be of a "latent nature and only become manifest when there is added to the primary operation a second operation upon, or a pathological process in, another region of the cortex." At present we are only able to say that the motor and sense areas are roughly determinable; their functions are not circumscribed, or, as von Monakow says, "*inselförmig*," but they are parts of a mechanism, and concerning their finer organization and their temporal and spatial relations to psychic phenomena we know almost nothing. Our present knowledge regarding cerebral-mental relations is not much greater than that of the neurologists of a hundred years ago. The facts at hand, as was pointed out several years ago, may warrant the conclusion that there is a "close connection between the brain and mental processes," but at the same time they also permit only the general conclusion that "the mental processes are not due to the independent activities of individual parts of the brain, but to the activities of the brain as a whole."

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REFLEX MECHANISMS AND THE PHYSIOLOGY OF NERVE[1]

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The present state of knowledge on the physiology of nerve is nowhere better presented than in the admirable Croonian Lecture (1912) of Lucas (21). The most important recent discoveries are here given in readable form, and it is shown that the Nernst theory of nerve-action, with some necessary extensions, is probably the beginning of a definitive theory. In the past year the two topics most actively discussed have been the production of reflex rhythms and the 'all or none' principle.

Sherrington, Brown, Forbes, Henkel, Vészi, and others have done a large amount of work on reflex rhythms which are produced by the nervous system independently of any rhythms which may be contained in the stimuli. The most common type of reflex found in a mammalian spinal preparation is that in which a single stimulation of an afferent nerve-trunk on one side causes the contraction of an ipsilateral flexor muscle, inhibition of the antagonistic ipsilateral extensor muscle, contraction of the symmetrical contralateral extensor muscle, and inhibition of the corresponding contralateral flexor (Sherrington, 30, p. 265): *i. e.*, the stimulus causes ipsilateral flexion (noci-ceptive reflex) and contralateral extension. In this arrangement lies the secret of motions of progression—step, walk, etc. Antagonistic muscles are regularly innervated reciprocally, symmetrical muscles are frequently but not invariably innervated reciprocally, while synergic muscles receive generally an identical reflex innervation. If, now, when a unilateral stimulation

[1] For last previous reviews in BULLETIN, see "Reflex Action": R. S. Woodworth, 1911, 8, 126–129; and "The Physiology of Nerve": E. B. Holt, 1913, 10, 146–151.

to afferent nerve has set up ipsilateral flexion and contralateral extension in two symmetrical limbs, the symmetrical afferent nerve of the opposite side be stimulated, the new stimulus commonly breaks through the effect of the old, the flexed limb is extended and the extended limb is now flexed—a step has been taken. The results attained by similar compounding of stimuli have been discussed at length by Sherrington (30, 31, 32), Brown (6, 7, 8, 10, 11, 12), and Forbes (13, 14). The striking point now is that if two opposing reflex stimuli, as above described, are given continuously and simultaneously (faradic stimulation) each reflex will alternately inhibit the other, and a rhythmical movement of progression results. How now, does this alternating inhibition arise? Baglioni (3) has suggested that as a limb flexes, afferent impulses (proprio-ceptive) are set up in the contracting muscles, and that these impulses inhibit the movement of flexion, thus allowing the contralateral stimulus that makes for extension to come into play. This, however, seems not to be the case, since these reflex rhythmical progressions are readily obtained in de-afferented preparations (Brown, 7, 10).

It has long been Sherrington's view that inhibition depends on a state of tissue "that at some restricted *locus* breaks or bars the transmitting power of the conductor." It has its seat "probably about the starting-point of the 'final common path'" (30), *i. e.*, the motor roots of the cord. In harmony with this Brown writes as follows regarding the origin of reflex rhythmic progressions (7, p. 286). "The cell-bodies and their processes of the efferent neurones of the antagonistic muscles form centers which mutually inhibit each other. A stimulus which falls upon one will therefore through it inhibit the other. But if this inhibition reduce the activity of the second center, that will inhibit the first less, and so the process will proceed until there is a limit set to this 'progressive augmentation of excitation.' . . . The limit may be set by a process of inhibitory fatigue. If this [progressive augmentation of excitation] proceeds the balance will turn in the opposite direction, and there will be a progressive augmentation of excitation in the other center until it too reaches its limit, when the process will set in in the other direction again. In such a scheme there is, however, no explanation of the occurrence of inhibition before excitation in time. It is not difficult to overcome the difficulty by postulating a pair of interposed centers between the afferent neurone and the efferent centers, and by supposing that these too mutually inhibit, and that, in addition, they inhibit the [p. 287] crossed primary centers. It

will be observed that in this scheme no explanation of the nature of the inhibitory process is given." (Precisely such a pair of "interposed centers" as Brown postulates is shown, on quite independent grounds, to exist, by Verworn, **36**, and Satake, **29**. The reviewer does not see how it solves the difficulty.) The essential feature of this view lies in the "inhibitory fatigue," which Sherrington and Forbes (**14**) locate at synapses.

A somewhat different view of reflex rhythms is given by Forbes (**13**, p. 297), who compares the breaking through of either of two opposed influences to "the stream of air emerging from a tube under water. A source of energy tends to produce an increasing force (*A*) opposing a relatively constant force (*B*) which tends to keep the energy pent [p. 298] up or potential; when (*A*) becomes greater than (*B*) the accumulated energy is released and becomes kinetic. The force (*B*) need not be constant, but if it increases during the increase of (*A*), its rate of increase must fall off below the rate of increase of (*A*) before the release can occur. The crucial point is this: that when once the release of energy begins it proceeds until more energy is released than is represented by the excess of (*A*) over (*B*). The condition which determines this may be figured as a sort of momentum of discharge, although it may not involve inertia in any strict sense of the word. . . . To develop rhythm of discharge there must be an approximation to the 'all or none law' of the heart beat. . . . There seems to be in the discharge of reflex impulses, and perhaps in all vital activities, some phenomenon akin to the 'all or none law' with its refractory period, which makes for intermittence of response." This shows a change of emphasis from the fatigue emphasized by Brown, to a "momentum of discharge" and "some phenomenon akin to the 'all or none law' with its refractory period." But it may be questioned whether these last two are at all identical functions. The possibility of explaining reflex rhythms by means of the principle of refractory period will be discussed later in connection with inhibition itself. A few further points in the papers so far mentioned remain to be noticed.

In his "Integrative Action of the Nervous System"[1] Sherrington described the "drainage theory" of McDougall, and one could infer that he deemed it a theory worth considering, although he by no means definitely espoused it. In the papers of the present year Sherrington makes no mention of this theory, although it is close to his topic: and Brown (**7**) declares directly that the "drainage

[1] For a review of this book see this BULLETIN, **8**, 119–125.

theory" is untenable. And indeed the papers of Sherrington, Brown, and Forbes yield considerable evidence that such is the case.

For many years it has been deemed an assured fact by most German physiologists that in ordinary life activities antagonistic muscles contract simultaneously, that indeed any nice precision of movement (whether voluntary or involuntary) could be secured only in this way. And it is of course certain that antagonists can be voluntarily contracted at the same time, and that in spinal preparations antagonistic muscles are often simultaneously innervated (*e. g.*, a degree of antagonistic innervation is characteristic of the "scratch reflex"). Yet it has not been made clear heretofore, so far as the reviewer knows, how Sherrington related these facts to his principle of "reciprocal innervation." (And here it is amusing to note that Descartes was aware of "reciprocal innervation," and that he clearly entertained the "drainage theory: "cf. "Les Passions de l'âme," article 11.) Sherrington has now touched on this matter (30, p. 269 ff.; 6, 31), stating explicitly that simultaneous reflex contraction of both members of a pair of antagonists is very common in every-day life and is to be observed experimentally. The stimulation of a single afferent nerve-trunk regularly gives reciprocal innervation of two antagonistic muscles; but if symmetrical right and left afferents are simultaneously stimulated each member of the pair of antagonists receives an excitatory *and* an inhibitory influence, and if the stimuli are properly adjusted as to intensity both of the antagonistic muscles may be contracted simultaneously. In other words "double reciprocal innervation" may produce "some degree of contraction by both the antagonists at the same time"; and thus the compounding of stimuli often masks the more fundamental function of reciprocal innervation. Furthermore, "the accuracy of a muscular contraction, delicately adjusted to the extent and force of the movement which is required, is usually a result of the graded combination of both inhibitory and excitatory influences coalescing upon the motor centers involved" (30). This is quite the German view. On the other hand Sherrington intimates that grace of movement and, say, abandon are probably found where each of the opposed muscle-groups is in turn completely at rest.

Now these facts somewhat modify the view of reciprocal innervation which a reader may easily have derived from the "Integrative Action." Firstly, antagonistic muscles are susceptible of simultaneous contraction; secondly, one muscle can actually receive

excitatory and inhibitory influences simultaneously, and this condition results not in a mere algebraic summation of the influences, but in a peculiar and characteristic muscle "tremor" (Sherrington, 32; Forbes, 13); and thirdly, both phenomena are functions of the spinal centers, and probably, in fact, of the motor half-centers. This shows that the inhibition which underlies reciprocal innervation is not the absolute block which some of Sherrington's earlier papers may have led readers to imagine. In fact a slight and rapid muscle tremor is regularly produced by the simultaneous play of excitation and inhibition on one muscle, just as "progression" and other rhythmic movements are regularly produced by the simultaneous play of excitation (and inhibition) on antagonistic muscle pairs. The problem now remains whether the inhibitions and other phenomena of "reciprocal innervation" and of "double reciprocal innervation" can be explained in terms of fatigue and recuperation (Brown), or of "momentum of discharge" or "refractory phase" (Forbes).

However these phenomena are to be explained, there seems to be little doubt (Baglioni, 3, to the contrary notwithstanding) that their mechanisms lie within the spinal levels and not far from the motor roots. Sherrington (30), Brown (7, 10), Forbes (14), Satake (29), Beritoff (4), Vészi (38), and Henkel (17) all emphasize this point. The last two investigators, on the basis of work with the strychnine-poisoned cord, speak of an "autonomous" rhythm of the nerve-ganglion.

Baglioni (3) gives a general discussion of theories of inhibition, with special reference to the theories of Hering, v. Cyon, McDougall, Verworn, and Fröhlich. The general tendency of the year's work seems to be toward the theory that nervous inhibition is due to the *interference* of nerve-impulses—a view that is not distinctly novel, but one which has received support and enhanced definiteness from recent work (with Einthoven's galvanometer) on the periodicity of the nerve-impulse, and on "refractory phase." The string-galvanometer has revealed oscillations in the nervous impulse of frequencies too high to be recorded by the older and less sensitive instruments. Furthermore, each oscillation appears to have its own refractory phase. And these two facts seem to imply that important interference phenomena are bound to appear wherever nervous impulses meet in converging toward their "final common path," *i. e.*, a motor nerve-root. Direct evidence of such interference of impulses, and evidence that is free from complication

with the obscure phenomena of synapse or muscle, is afforded by the Wedensky effect. This is the phenomenon that "at a certain stage in the fatigue or narcosis of a muscle nerve preparation, a series of strong or rapidly recurring stimuli may produce a small initial contraction only (Anfangszuckung), whereas a series of weak or slowly recurring stimuli produce a continued tetanus." In an ingenious and admirable paper (**2**) Adrian studies this phenomenon without making use of the muscle or the myo-neural junction: the effect exists in the plain nerve-fiber. And he corroborates Lucas's explanation (**22**) that the Wedensky inhibition is due to the fact that the second or any subsequent stimulus finds the nerve incompletely recovered from the just-previous excitation, *i. e.*, in refractory phase, so that the second or subsequent propagated disturbance is by just so much reduced. The strength and frequency of the stimuli can be so adjusted that all propagated disturbances after the first will be too weak to cross the myo-neural junction. Here then we have inhibition by *interference* within plain conducting fiber. The possible complications where nerve-impulses of different (and perhaps inconstant) periodicities converge on common paths cannot be foreseen: but it is clear that inhibition by interference is an hypothesis that may reasonably be entertained in connection with the rhythms produced by double reciprocal innervation. It is probably this which Forbes has in mind when he states (**13**) that it is apparently "some phenomenon akin to the 'all or none law' with its refractory period, which makes for intermittence of response."

But if reciprocal innervation and, as we shall later see, the "all or none law" are frequently masked by the necessity of stimulating whole nerve-trunks instead of single nerve-fibrils and of observing the results on entire skeletal-muscles instead of on a single motor fibril and its attached muscle elements, the principle of inhibition by interference is still more deeply hidden in the network of the spinal cord. For this reason although the interference of nerve impulses is coming to be frequently and confidently spoken of, as by Verworn (**36**), Satake (**29**), and Vészi (**38**), very little that is definite has so far been brought out. Baglioni (**3**) opposes the theory of inhibition by interference. Some technique is urgently needed by which the activity of a single nerve-fibril can be observed and then followed.

Any very conclusive work on the periodicities of the nerve-impulse is also thwarted by the necessity of observing the resultant

oscillations from a mass of nerve-fibrils simultaneously excited. Verworn states (36) that the frequency of oscillation depends on the freshness and vigor of the nerve. Mines (26) observes in muscle electrical responses at the rate of 50 per sec. (the rate of the faradic stimulus) while the mechanical response (contraction) shows no fluctuations. Henkel (17) states that the action-currents in nerve and muscle are rhythmical, and that strychnine cramp is due to an "autonomous" rhythmic discharge of the ganglion centers. The most important discovery is that of Fröhlich (16) who finds that the action-current, in the cephalopod eye under stimulation by light, is oscillatory, and that the frequency varies with the intensity and *wave-length* of the stimulating light. The frequencies range from 20 to 90 per sec. He states that these excitations, varying in period with the wave-lengths of the stimulating lights, "are to be looked on as the physiological basis of color-sensation." This is a definite hint as to the nature of "specific nerve energies," and it further complicates the problem of interference. It should be mentioned, finally, that if the inhibition of nerve-impulse is due to interference, this inhibition would be chiefly manifested where nerve-impulses meet, *i. e.*, at the synapses: and this would accord with Sherrington's view that the synapses are the seat of inhibition. There might be some question, too, as to how far the "fatigue" of synapses is an interference phenomenon (Forbes, like Sherrington, places fatigue at the synapses: 14).

No work of the year shows more resourcefulness and elegance of method than the several papers on the "all or none law." The law is generally confirmed. Adrian, in two remarkable papers (1, 2), shows that a propagated disturbance in nerve on emerging from a region of decrement (*e. g.*, a narcotized or asphyxiated tract) recovers its full intensity, and this within at most 5 mm. from the point of emergence. "It is extremely probable that the magnitude of the disturbance which has travelled a few mm. in normal tissue will be independent both of the strength of the stimulus which has set it up and of any changes which it may have undergone in its course down the nerve before it entered the normal part." "The recovery of a disturbance . . . is not at all affected by the extent of the reduction it has suffered, provided that this is not great enough to extinguish it completely." The "all or none" principle is often veiled in work with nerve-trunk, where the number of elements stimulated is variable. Threshold stimuli applied to a nerve-trunk excite only a portion of its fibrils (Lodholz, 19).

In passing through a region of decrement the nerve-impulse suffers a continuous diminution, and if the region is long enough it will be extinguished (Adrian, 1, 2; Lucas, 21, 22), and the distance required to extinguish it "is the same whether the impulse originates from a very weak or a very strong stimulus" (Lodholz, 19). The impulse decreases most rapidly when it first enters the region of decrement: its decrease is an exponential function of the length of impaired nerve that it has traversed. This decrease is, however, per linear unit traversed a logarithmic function of the time during which the region of decrement has been exposed, say, to asphyxiation, *i. e.*, of the depth of narcosis (Lodholz, 20). The speed of propagation is at the same time reduced; and both phases of the action-current suffer the same retardation. Beyond the region of decrement the impulse resumes its original rate of propagation (Fröhlich, 15). If Tait's theory that the absolute refractory state corresponds to the rising phase of the electrical response, the last statement quoted from Fröhlich would *seem* to be at variance with the observation of Lucas (22), that "at a stage of its action at which alcohol has made a nerve conduct with a large decrement, the time of recovery (refractory period) is not prolonged."

The fact of summation of nerve stimuli bears interestingly on the all or none principle. The most interesting theory of summation is that of Lucas (21) who gives evidence for a preliminary process of local change, or "local excitatory process" which precedes the propagated disturbance. Summation takes place wholly in the local excitatory process, for in summation phenomena the earlier (subliminal) stimuli do not seem to set up any propagated disturbance whatsoever. The "electrical response" of nerve is essentially connected with the propagated disturbance alone. On the other hand Lucas (22) gives an interesting reinterpretation of Grünhagen's gas-chamber experiment, showing that irritability and conductivity do not vary independently. "It appears probable that the need for an increase of current-strength [exciting stimulus] and the decrement in conduction arise from a common cause, an increased difficulty in setting up the propagated disturbance." Mines discusses the phenomenon of Treppe and of the summation of contractions in tetanus (26), arguing that they are not at variance with the all or none principle in either nerve or muscle. In other cases, as in pulsus alternans (27), an apparent summation is produced by the varying number of fibers which are at different times set in action. This is especially the case as a stimulus is gradually increased from threshold strength (Lodholz, 19).

The all or none law is thus upheld by Verworn, Lucas, Adrian, Lodholz, Vészi, Mines, and others. Brown offers some experimental evidence against it (9), but he admits that the evidence is not wholly conclusive. Adrian is of opinion that the only way to produce a sub-maximal propagated disturbance is by timing the stimulus to fall on the partially refractory stage of a previous disturbance (2): Vészi seems to be of the same opinion (38). It is to be noted that Verworn (36) attributes that *Bahnung* which is the basis of habit-formation, *i. e.*, the establishment through repeated use of reflex patterns, to "hypertrophy" of the paths so used. This is also the physiological basis of memory.

As to the more precise nature of nerve-action, the modified Nernst theory seems to be gaining ground (Lucas, 21). Lillie (18) traces interesting parallelisms between the lowered toxic actions of various salt solutions, their lowered power to increase the permeability of plasma-membranes of irritable tissue, and their lowered stimulatory power in the presence of anæsthetics. Thus in anæsthesia "an anti-cytolytic or antitoxic action" goes "with the anti-stimulating action of the anæsthetic." Traube (34) adduces important considerations relating the narcotic effects of substances which are soluble in water with the surface-tension of the resulting solution. "By accumulating at the boundary between cell-walls and (the contained) cell-fluid, narcotic substances reduce the electric contact-potential, and so operate directly to inhibit the propagation of motor and sensory stimuli by the nerve-centers." Traube believes that narcotics penetrate the nerve-cell: Menten (25) reports that potassium salts "in anæsthesia produced by lipoid-solvent anæsthetics" penetrate medullated nerve-fiber. Mansfeld and Bosányi (23) report that in magnesium-narcosis the magnesium does not penetrate the nerve-cell: it affects the cell-membrane. Porter (28), studying reflex phenomena under asphyxial conditions, states that "the threshold of the nerve-muscle preparation remains unaltered during even severe asphyxia"(!). Mares (24) states that the process of death in nerve-tissue presents a phase in which Pflüger's law of stimulation is reversed, *i. e.*, the excitation is at the anode when the current is closed; at the cathode when the current is broken. Verzár (37) finds that the passage of an action-current over a nerve diminishes both of the extra-polar polarization-currents: this probably comes from a change in permeability of the semi-permeable cell-membrane. Stübel (33) finds microscopically visible changes in the myeline sheath of nerve due to protracted or even

brief stimulation. An interesting theory of the functions of the eye, in general agreement with the Nernst theory of nerve-action has been proposed by Troland (35). Lastly, Boeke (5) seems to have demonstrated conclusively that in some cases at least the central end of a motor nerve (hypoglossus) if joined to the peripheral stump of a sensory nerve (lingualis), will regenerate along the path of the latter. Some of the motor fibers, arrived at the surfaces of the tongue, actually penetrated taste-buds, and other epithelial cells. Other fibers seemed to try to form new motor end-plates.

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REPORTS

Eleventh Annual Conference of Experimental Psychologists.

The Annual Conference of Experimental Psychologists was held at the psychological laboratories in Schermerhorn Hall, Columbia University, on Thursday and Friday, April 9 and 10. About forty psychologists were present, representing the laboratories of California, Cincinnati, Clark, Columbia, Cornell, George Washington, Georgia, Harvard, Minnesota, Pennsylvania, Princeton, Vineland, Wesleyan, Wisconsin, Yale, Bureau of Education of Chicago, Brooklyn Training School, and the Abbe Laboratory of Cleveland.

In reporting the work in progress in the various laboratories the Psychological Index classification of topics was followed, and all work bearing on a given topic reported under that heading. This was supplemented by more general reports from laboratories which were not represented at the earlier sessions. This method brought out interesting indications as to the topical distribution of current research work in the laboratories represented. Perhaps the most striking fact is the general neglect of sensation, psychophysics, and anatomy and physiology of the nervous system. The larger number of reports bore on such topics as learning, memory, tests, mental defect, work, attention, judgment, association, and affective experiences.

The social features included a dinner and smoker given by the department of psychology of Columbia, at the Faculty Club. Two forenoon and two afternoon sessions were held and opportunity was given for visiting psychologists to inspect the laboratory and to take part in several researches there in progress.

H. L. Hollingworth

NOTES AND NEWS

THE EDITORS of the PSYCHOLOGICAL REVIEW PUBLICATIONS take pleasure in announcing the election of Dr. Shepherd I. Franz to the editorship of the PSYCHOLOGICAL BULLETIN. Professor Franz will take charge beginning with the May issue.

THE present number of the BULLETIN, dealing especially with the physiology of the central nervous system, has been prepared under the editorial care of Professor Roswell P. Angier.

WE REGRET to announce the death on February 16 of Dr. Theodate L. Smith of Clark University, the author of many contributions to child psychology. Also the death on April 9 of Professor Alexander F. Chamberlain, of Clark University, author of "The Child and Childhood in Folk-Thought" and of many contributions to anthropology.

A CONFERENCE on individual psychology was held under the auspices of the department of psychology at Columbia University April 6–8. Papers were read by a large number of former students of the department.

DURING the current semester, Professor W. V. Bingham is on leave of absence from Dartmouth College for travel and for study at Cambridge University. He will return to Hanover in season to take charge of the Dartmouth summer session, of which he is director.

THE psychological laboratory at Wellesley College was completely destroyed by the burning of College Hall on March 17. Nearly all the apparatus was lost, besides several valuable records. The MS. of a thesis on the Application of the Method of Right and Wrong Cases to Intensive Smell Discrimination by Miss Alice Forbes was consumed; also a much more extensive piece of work upon a problem in Free Association, and a series of intelligence-test records made upon 100 members of the college, 40 normal children and a number of defective children at Waverley. Professor Mary W. Calkins was sleeping in the building when the fire broke out, and had a narrow escape.

THROUGH the kindness of relatives the psychological library of the late Professor Arthur H. Pierce has been presented to Smith College.